EVALUATION OF STUDIES ON HUMAN EXPOSURE TO SOIL LEAD RESIDUES

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1.0. INTRODUCTION AND SUMMARY

The purpose of this report is to critically review three technical articles concerning soil lead exposure, transmitted from Kenneth Miller of the Illinois Environmental Protection Agency to Stephen Holt of NL Industries, Inc. on October 26, 1989 in reference to the remediation of the Granite City/Taracorp Superfund site in Granite City, Illinois. Pertinent information obtained in a recent updated review of the literature on the relationship between soil lead residues and lead exposure in children is also included in this report, and which was used to develop a soil lead remedial objective.

In summary, the reports pertaining to vegetable uptake of lead do not appear to be directly relevant to the derivation of soil lead remedial objectives for the Granite City/Taracorp site, and the information provided on lead exposure of children from contaminated soil is drawn from a biased sample of outdated studies on the subject. Review of recent information on lead exposure published after 1980 indicates substantial decreases in baseline lead exposure and the incremental contribution of soil lead residues to lead exposure, due primarily to the phasedown in leaded fuels and other lead uses. Analysis of this recent information indicates that lead remedial objectives for soil of 1,000 ppm at the Taracorp/Granite City site are both protective and technically defensible.

2.0 REVIEW AND EVALUATION

2.1. Concerning the Bassuk (1986) report on the uptake of lead in lettuce, the study was conducted with $PbCl_2$, a soluble lead salt which will undoubtedly be taken up by plants to a much greater extent than weathered environmental lead soil residues which are composed of oxides, sulfides, soil-bound, and other such insoluble and much less bioavailable forms of lead (as reviewed in the original RI report and in U.S. EPA, 1989). As a result, this data has no relevance to assessment of lead exposure in the Granite City study area.

2.2. The Spittler and Feder (1979) paper summarizes a study which attempted to correlate lead uptake from urban soils into various vegetable types. The results showed the highest level of uptake show a clear relationship between lead uptake and lead soil concentration, with lettuce giving the highest uptake levels. However, even with lettuce, the level of uptake was comparatively low: approximately 5% of the concentration in the soil on a dry weight basis. This avenue of lead exposure was addressed in the risk assessment prepared for the Granite City RI by considering soil exposures for an individual during the period of adulthood, as well as for childhood outdoor activities. This would constitute an upper bound lifetime chronic exposure rate upon which to estimate health risks from various levels of soil lead residues. One justification given for the assumption of adult exposure was the possibility of gardening with accompanying ingestion of soil lead residues adhering to the hands, vegetable crops, or both. Such exposures are higher than would be expected via ingestion of lead taken up into home-grown vegetables. It is interesting to note that the lead levels in the Boston garden soils studied were to a large extent comparable with the levels noted in the Granite City study area, even in the absence of lead smelting.

While consumption of lead-contaminated vegetables may present an avenue for lead exposure, the derivation of soil cleanup objectives for lead at the Taracorp/Granite City site should remain focused upon children. The reasons for this include the behavior of children which enhances the transfer of soil to the mouth, the incompletely developed nervous system, and increased lead absorption rate relative to adults. The importance of these factors in determining the principal group of concern for exposure and health effects of lead is evidenced by the voluminous literature base developed on the topic, as cited in the following evaluation.

2.3. The following is a discussion and analysis of the key elements contained in "Lead in Soil: Recommended Maximum Permissible Levels", published in Environmental Research, 49: 136 - 142 (1989), by S. Madhavan, K.D. Rosenman, and T. Shehata. Citing sources contained in a 1980 review of the literature conducted by M.J. Duggan assessing human exposure to lead in urban dust and soil, Madhavan et al. suggest a protective soil cleanup objective of 600 ppm. This conclusion is invalid for several reasons.

A LOWEST OBSERVED EFFECT LEVEL FOR LEAD IS IMPROPERLY DEFINED:

A lowest observed adverse effect level (blood concentration) for lead in humans is identified by Madhavan et al. as 10 ug/dl. This was associated with inhibition of the enzyme ALAD, a key enzyme in the biosynthesis of heme. However, this inhibition is translated into decreased hemoglobin levels and anemia only at substantially higher blood lead levels (40 to 80 ug/dl from a number of investigations reviewed in ATSDR, 1988). Thus, ALAD inhibition at 10 ug/dl should be viewed as a sensitive biological indicator of lead exposure, rather than an overt adverse effect at these lower blood lead levels.

A VALID ALTERNATIVE EXPOSURE ASSESSMENT METHOD IS INCORRECTLY REJECTED:

Madhavan et al. state that data on estimates of the amount of soil ingested by children show a 100-fold variation and thus are not useful in deriving a "safe" soil level for lead. Therefore, Madhavan et al. use information only on the relationship between blood lead concentration and soil concentration to derive their criterion. However, the cited sources show good consistency in estimated soil rates (EPA Exposure Factors Book, 1989). Both the Binder et al. (1986) and Clausing et al. (1987) studies directly measured children's soil ingestion in controlled experiments, and show less than a two-fold variation in mean daily soil ingestion rate (127 - 230 mg/day). Thus, an additional approach to lead exposure analysis was rejected incorrectly, even though U.S. EPA (1989) successfully used such an approach in developing its validated biokinetic lead exposure model.

STUDY SELECTION METHOD WAS BIASED AND AN INVALID DATA POINT IS INCLUDED IN THE ANALYSIS:

Duggan (1980), a compilation and review of the studies on blood lead and soil exposure conducted primarily before 1975, was used by Madhavan et al. as the source of 21 derived soil and/or house dust lead/blood lead correlations. In Duggan's analysis, 19 of the 21 studies, with correlation slopes for the contribution of soil and/or house dust lead, ranging from 1.6 to 14 ug/dl per 1000 ppm soil lead (some of which represent averages of replicate studies within a single cited source), which showed a statistically significant difference in the range of soil lead concentrations measured, were used to derive an estimated increase (both arithmetic mean and median) of the order of 5 ug/dl per 1000 ppm of soil or dust lead.

Madhavan et al. selected only 8 of the 21 individual blood lead/soil lead correlation estimates, ranging from 0.6 to 65.0 ug/dl per 1000 ppm, from the Duggan (1980) compilation for their analysis, to isolate uptake in children less than 12 years of age ("... the most susceptible group to lead toxicity"...) and to eliminate the influence of other sources of lead exposure (house dust was cited). No other justification was provided for the selection of these eight values. In fact, Duggan (1980) note that there was no clear separation of the slope values seen in soil studies vs. house dust studies. This opinion was confirmed by U.S. EPA (1989). Thus, the basis for study selection in the Madhavan et al. analysis is questionable, particularly the exclusion of house dust studies, and this

diminishes the statistical confidence of the resulting estimate of slope. Madhavan et al. determined a geometric mean (based on an assumption of lognormal blood lead distribution) for the 8 studies taken from Duggan (1980) of 3.41 ug/dl per 1000 ppm soil lead with a geometric error of 1.75 ug/dl. An upper bound 95% confidence limit of 8.5877 ug/dl per 1000 ppm is reported by Madhavan et al..

Examination of the table in Duggan (1980) from which the 65.0 ug/dl per 1000 ppm value (from the Angle et al. reference) was selected by Madhavan et al. for inclusion indicates that the soil lead residue range was considerably less than 1000 ppm (97 to 219 ppm), and that the variation was not considered statistically significant. Thus, this value cannot be considered a "slope" describing the incremental contribution of increasing levels of soil lead to blood lead, as stated by Madhavan et al.. Instead, it represents only an estimate of blood lead obtained by extrapolation from a single soil lead level typical of urban background levels to a soil lead level of 1000 ppm. Derivation of a valid correlation slope requires that both variables be measured over a statistically significant range of values, preferably encompassing the entire range of interest. As a result, it is inappropriate to include the value of 65.0 ug/dl per 1000 ppm in the statistical treatment of estimated slopes, because it is not a slope. U.S. EPA (1986) did not include this value in its analysis of soil lead uptake in children. Furthermore, 65 ug/dl of children's blood lead represents a potential frank effect level for lead toxicity in children for effects including anemia and neurotoxicity (ATSDR 1988, CDC 1985). Such readily observed toxicity purported by Madhavan et al. to be associated with soil lead levels of 1000 ppm is not consistent with public health investigations conducted in Granite City (as reviewed in the Granite City RI report), which did not reveal elevated lead exposure or clinical manifestations of toxicity, nor with other reviews (including CDC 1985 and EPA 1986). This provides an additional basis for exclusion of the 65 ug/dl value from derivation of soil/blood lead correlations.

Excluding the highest value in the data set (65.0 ug/dl per 1000 ppm) because it does not represent a slope of the blood lead/soil lead relationship from the calculation reduces the 95% upper confidence estimate of the slope to 4.52 ug/dl. This would correspondingly increase the maximum permissible soil lead level derived by the Madhavan et al. approach to 1200 ppm.

LEAD UPTAKE IS ASSUMED WITHOUT SUPPORTING DATA TO BE LINEAR WITH CONCENTRATION:

Madhavan et al. present a table which assumes a linear relationship between blood lead and soil lead down to 1 ug/dl and 116 ppm. A basis for this assumption of linearity is not provided. In fact, in citing the Centers for Disease Control (CDC, 1985) review of some of the same information utilized by Duggan (1980), Madhavan et al. appear to contradict their own assumption of linear uptake. Specifically, CDC concludes: "In general, lead in soil and dust appears to be responsible for blood lead levels in children increasing above background level when the concentration in the soil or dust exceeds 500-1000 ppm.". This statement clearly suggests that soil lead of less than the 500 to 1000 ppm range does not result in observable blood lead increases. Choosing 5 ug/dl as a "tolerable" level of

blood lead to be added to baseline blood lead, Madhavan et al. present the associated value of 600 ppm of soil lead from their analysis as a protective level, adding this incrementa blood lead increase to 1976 - 1980 baseline blood lead medians of 16 and 20 ug/dl for white and black children, respectively. Recently, however, U.S. EPA (1989) reviewed the continuing trend in decreasing blood lead in children due to phasedown of leaded gasoline and other diminishing sources of lead exposure, and has determined that 1990 blood lead values in children will be of the order of 5 ug/dl. The basis of this determination is discussed in the following section.

THE DATABASE ANALYZED IS NOT RELEVANT TO CURRENT CONDITIONS:

In addition to the flaws noted above, the Madhavan et al. study relies entirely upon older, generally pre-1975 data, thus excluding such important contributions as the Stark et al. (1982) study, which was conducted with large numbers of children in an urban U.S. population (including blacks, generally thought to accumulate lead to a greater degree than other races) in the absence of active smelting or other comparable industrial sources. In addition, Rabinowitz and Bellinger (1988), and U.S. EPA (1989) both contain more recent data strongly suggesting that the contribution of soil lead to children's blood lead may be substantially less than indicated in Duggan's pre-1980 data set. These contemporary data are highly relevant to remediation of soil lead residues at the Taracorp/Granite City site and are discussed further below.

Madhavan et al. also utilized outdated information on the baseline levels of blood lead in children, citing "median" blood leads with current geometric mean blood lead levels of 16 ug/dl (white) to 20 ug/dl (black) children, 6 months to 5 years of age. As is well reviewed in U.S. EPA (1989), lead exposures have been declining rapidly in recent years, due not only to the phasedown of leaded gasoline, but also to the elimination of the use of leaded solders in metal food containers, and the replacement of water distribution systems containing leaded solders. For example, mean dietary lead exposure in children was estimated to have decreased from 52 ug/day to 8.8 ug/day between 1978 and 1990. This report, which was reviewed and approved by the U.S. EPA Clean Air Scientific Advisory Committee, estimated, through the use of a validated biokinetic lead exposure model and the 1978 NHANES II blood lead data, decreases in children's blood lead due to phasedown of leaded gasoline of 8.6 ug/dl, decreases in blood lead due to decreased dietary lead exposure of 0.9 to 1.8 ug/dl, and decreases in maternal lead exposure producing decreased blood lead of 0.2 to 0.3 ug/dl. As a result, blood lead levels of 2 year old children in 1990 will average (geometric mean) from 4.2 to 5.2 ug/dl (compared with the average 1978 value of 14.9 ug/dl), and also from 3.5 to 5.8 ug/dl in adults (down from average values of 10.8 to 17.7 ug/dl).

Several recent studies are available to make more accurate inferences relative to Madhavan et al. on the contemporary relationship between childrens' lead exposure from soil, in light of the qualitative and quantitative changes in national lead sources of exposure. For example, Stark et al. (1982), conducted a study of the exposure of urban children to soil lead in the period from 1974 to 1979. U.S. EPA (1986) represented this

study as a good median estimate of the relationship between soil and children's blood lead, and Stark et al. (1982) was therefore used in the Granite City/Taracorp risk assessment on this basis. The study was conducted in New Haven, Connecticut, using 153 children of age 0 to 1 year, and 334 children of 2 to 3 years, and soil ranging in lead content from 30 to over 7,000 ppm. An analysis by U.S. EPA (1986) of the data in this study gave a slope estimate of 1.8 ug/dl blood lead per 1,000 ppm soil lead, approximately one-half of the Madhavan et al. mean estimate.

In addition, Rabinowitz and Bellinger (1988) conducted a similar study of a population of children in Boston during 1981. Using a sample size of 195 children aged 6 months to 24 months and a range of soil lead of 7 to 13,240 ppm, a slope of 0.89 ug/dl per 1,000 ppm soil lead was estimated. In addition, the population was divided approximately evenly into populations of children who were said to finger and hand mouth less, and those with more mouthing activity, as determined by a statistical analysis of psychologist judgments on the frequency with which the children placed their fingers, hands, or foreign objects in their mouths. The slope estimate for the less mouthing group was 0.57 ug/dl per 1,000 ppm (standard error of 0.2), and 1.6 ug/dl per 1,000 ppm of lead (standard error of 0.5) for the greater mouthing group. Because the study population did not live in crowded conditions which might enhance exposure to leaded paint residues in soil near houses, the authors caution that the slope might be steeper under more crowded, urban environmental conditions.

However, as reviewed in U.S. EPA (1989), Johnson and Wijnberg (1988) conducted a study commissioned by the Centers for Disease Control in 1983 of children living in the vicinity of the ASARCO lead smelter in East Helena, Idaho. These investigators derived a slope estimate of 1.4 ug/dl per 1,000 ppm lead, with a soil range of 158 to 1,549 ppm studied, once again less than one-half the mean estimate of Madhavan et al.. The data of Johnson and Wijnberg (1988) was one study used by U.S. EPA (1989) to successfully validate its mathematical biokinetic model predicting blood lead levels in various age groups based on uptake, absorption and elimination rates via several physiological compartments and exposure routes. Consequently, three recent empirical studies are available which indicate that the relationship between blood lead concentrations and soil lead ranges from 0.6 to 1.8 ug/dl per 1000 ppm.

2.4 DERIVATION OF A SOIL LEAD CLEANUP OBJECTIVE UTILIZING RECENT DATA

Thus, utilizing data from Stark et al. and Rabinowitz and Bellinger (1989), as well as estimates of current base-line lead exposure further supported by the CDC's ASARCO study, it is possible to utilize the approach of Madhavan et al to derive a clean-up objective for soil lead in Granite City. Rounding the slope of the Stark et al. (1982) and the Rabinowitz and Bellinger (1988) high mouthing behavior study group to 2.0 ug/dl per 1,000 ppm lead, and adding 1.0 ug/dl (two standard errors on the geometric mean of the Rabinowitz and Bellinger (1988) study), it appears that exposure of a child with high hand to mouth behavior to soil lead levels of the order of 1,000 ppm will add approximately

3.0 ug/dl to baseline blood lead as an upper bound estimate using contemporary data. In view of recent projections that the national mean baseline blood lead concentration in young children may be up to 5.2 ug/dl (geometric mean), an upper bound estimate of childrens' blood lead resulting from exposure to 1,000 ppm soil lead appears of the order of 8.2 ug/dl. This level is below the blood lead level of 10 ug/dl incorrectly cited by Madhavan et al. as a lowest observed adverse effect level, and considerably less than the 25 ug/dl represented by these authors to result from exposure to the 600 ppm maximum permissible soil lead level under worst case conditions. A margin of uncertainty of approximately 2 ug/dl or more thus exists between the upper bound blood lead estimate of 8.2 ug/dl for exposure to 1,000 ppm soil lead and the 10 ug/dl lowest observed effect level for ALAD inhibition. This will allow for protection of site-exposed individuals who are at the upper end of both the 1990 baseline blood lead distribution (estimates of the geometric standard deviation were not available for the current mean estimate) and soil lead uptake distribution from overt lead toxicity (as opposed to ALAD inhibition alone). In consideration of the fact that the baseline blood lead already contains a contribution from baseline soil exposure (U.S. EPA 1989), it is concluded that 1,000 ppm soil lead residues at the Taracorp/Granite City site will not represent a source of adverse health effects for the worst case exposure population.

2.5 CONCLUSIONS

In conclusion, Madhavan et al. (1989) conducted an analysis of a biased, outdated (over ten years old) data set which is of little relevance to conditions present at the Taracorp/Granite City site. Applying conservative assumptions to this analysis, Madhavan et al. conclude that 600 ppm soil lead represents a maximum permissible level where exposure of children is likely. The exclusion of more recent analyses, reflecting the decrease in base-line lead exposure and blood lead levels and decreases in the contribution of soil lead to base-line lead exposure, renders this analysis and conclusion invalid. Consideration of the more recent evidence, as well as the remainder of the site data base as incorporated into the Granite City/Taracorp Remedial Investigation provide ample basis for selection of 1000 ppm as a protective soil lead cleanup objective.

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